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Left posterior fascicular block, state-of-the-art review: A 2018 update

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ABSTRACT

We conducted a review of the literature regarding epidemiology, clinical, electrocardiographic and vectorcardiographic aspects, classification, and differential diagnosis of left posterior fascicular block.

Isolated left posterior fascicular block (LPFB) is an extremely rare finding both in the general population and in specific patient groups. In isolated LPFB 20% of the vectorcardiographic (VCG) QRS loop is located in the right inferior quadrant and when associated with right bundle branch block (RBBB) ≥40%.

The diagnosis of LPFB should always consider the clinical aspects, because a definite diagnosis cannot be made in the presence of right ventricular hypertrophy (RVH) (chronic obstructive pulmonary disease (COPD)/emphysema), extensive lateral myocardial infarction (MI) or extremely vertical heart.

Intermittent LPFBs are never complete blocks (transient or second degree LPFB) and even in the permanent ones, one cannot be sure that they are complete. When LPFB is associated with RBBB and acute inferior MI, PR interval prolongation is very frequent.

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1. Introduction

Left posterior fascicular block (LPFB) is an intraventricular conduction disturbance of the left posterior fascicle (LPF) of the left bundle branch (LBB), which travels to the inferior and posterior region of the left ventricle (LV), the left ventricle inflow tract (LVIT). In LPFB, there is a total or partial (transient or second degree LPFB) conduction defect of the supraventricular electrical impulse. Consequently, the stimulus is conducted to the LV via the left septal fascicle (LSF), which first activate the midseptal and apical region of the LV [1] and to the left anterior fascicle (LAF), which is directed to the base of the anterolateral papillary muscle of the mitral valve (ALPM), inserting into the upper lateral wall of the LV along its endocardial surface [2].

Typical electrocardiographic (ECG) and vectorcardiographic (VCG) changes induced by the three fascicular blocks are illustrated in Fig. 1.

The LPF is the least vulnerable division of the intraventricular conduction system. Rosenbaum et al. referred that the causes of greater vulnerability of the LAF compared to LPF are: anatomical (the LAF is smaller in diameter - 3 mm versus 6 mm - and shorter - 35 mm vs 30 mm); electrophysiological (as a consequence of its greater extension and smaller diameter, the depolarization and repolarization of the LAF is slower than of the LPF); vascular: LPF is irrigated by two coronary systems, from the left anterior descending artery (LAD) and the right coronary artery (RCA), and localization (topography): the LPF runs through a more protected area, the LVIT with less mechanical pressure impact. On the other hand, the LAF runs diagonally through the subendocardium of the left ventricular outflow tract (LVOT). This region is subject to a great turbulence and high pressure, which partially explains its greater vulnerability [3–6].

2. Epidemiology

Isolated LPFB is very rare finding (0.1% of all intraventricular conduction defects). Little data exists regarding the prevalence of LPFB. Haataja et al., in Finland, based on the Health 2000 Survey conducted in 2000/2001 studied 6354 individuals aged 30+. In this

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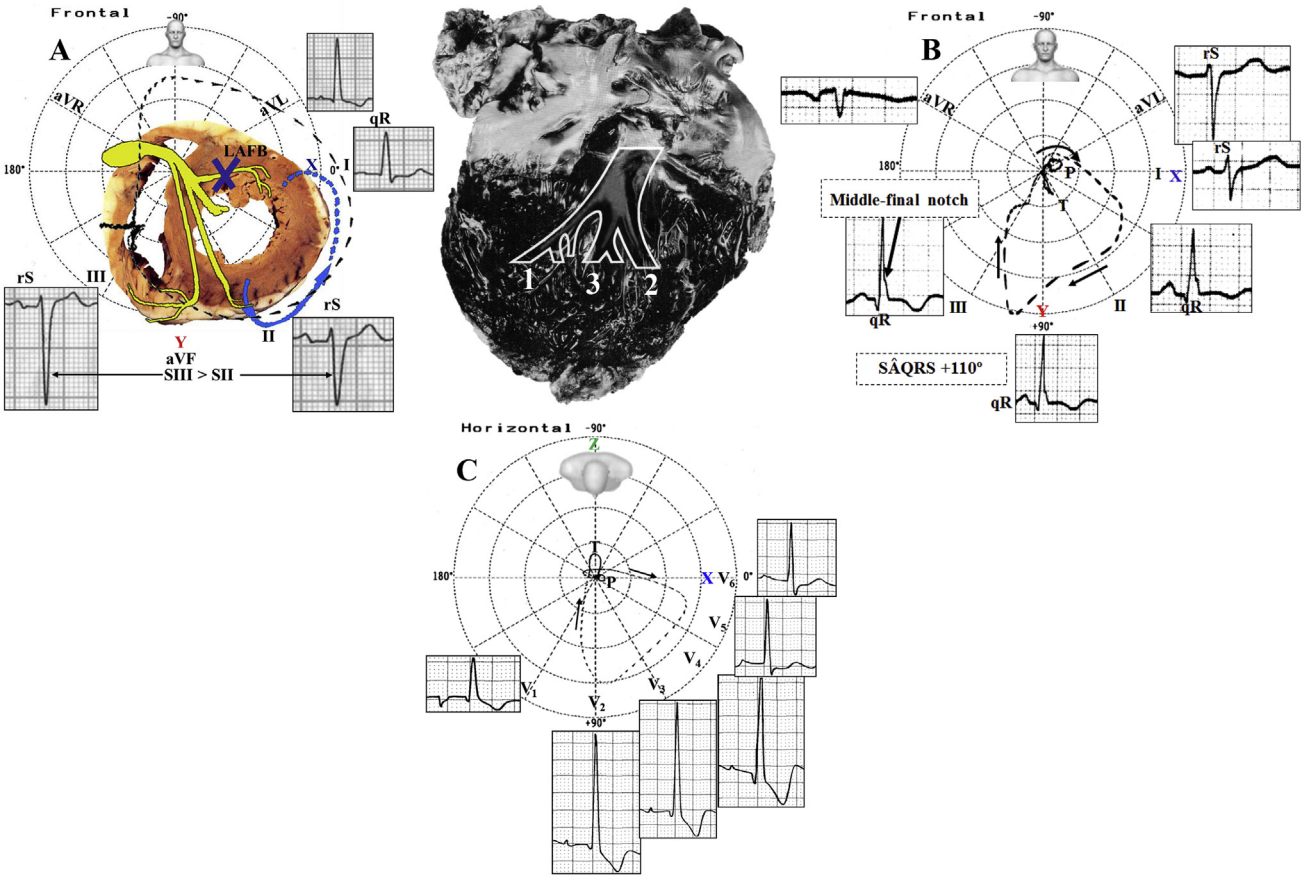


Fig. 1. LAFB (Fig. 1-A) causes left axis deviation in the frontal plane (usually between −45° and −90°); LPFB (Fig. 1-B) causes right axis deviation in the frontal plane (QRS axis ≈+120°, between +80 and +140°); while left septal fascicular block (Fig. 1-C) causes prominent anterior QRS forces in the horizontal plane.

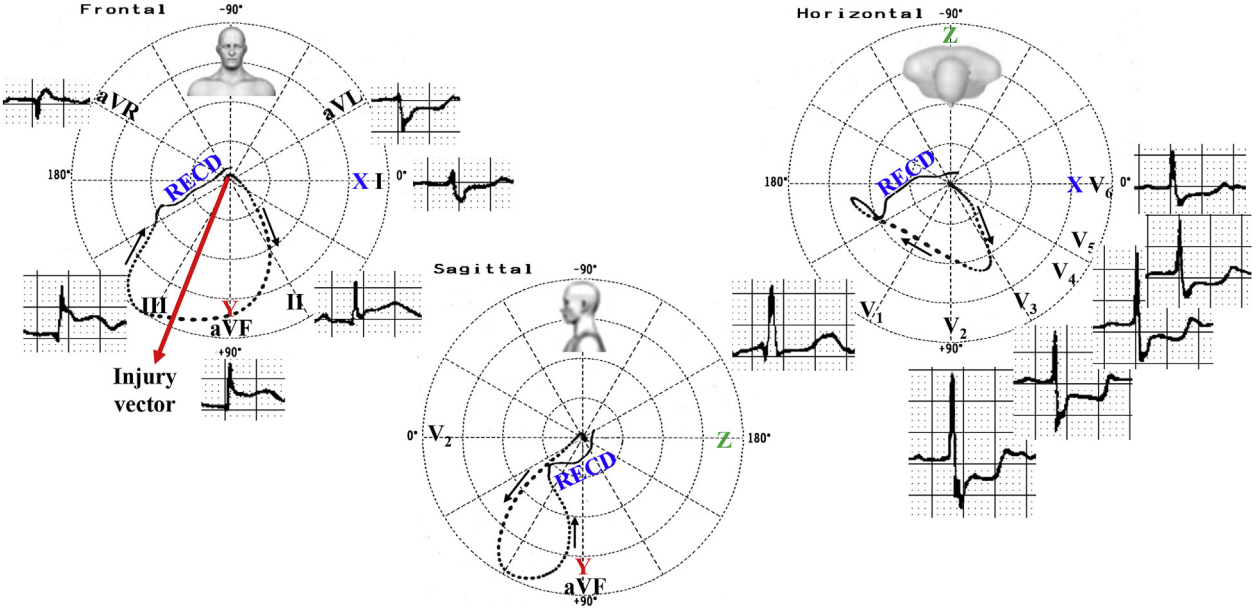


Fig. 2. Example of LPFB + complete RBBB Kennedy type III + hyperacute phase of inferior MI by obstruction of RCA.
FP: QRS axis +150°, VCG QRS loop with clockwise rotation and located in inferior quadrants (>40% area in inferior right quadrant), injury vector pointed to III suggesting proximal obstruction of RCA, ST segment depression in I and aVL, ST segment elevation (STE) in inferior leads (STEIII > STEII). HP: QRS loop located in the anterior quadrants, CW rotation, right end conduction delay (RECD) in the right anterior quadrant, complete RBBB Kennedy type III. LSP: all QRS loop located in anterior and inferior quadrants, QRS loop with counterclockwise rotation. Conclusion: LPFB + complete RBBB + acute inferior MI.

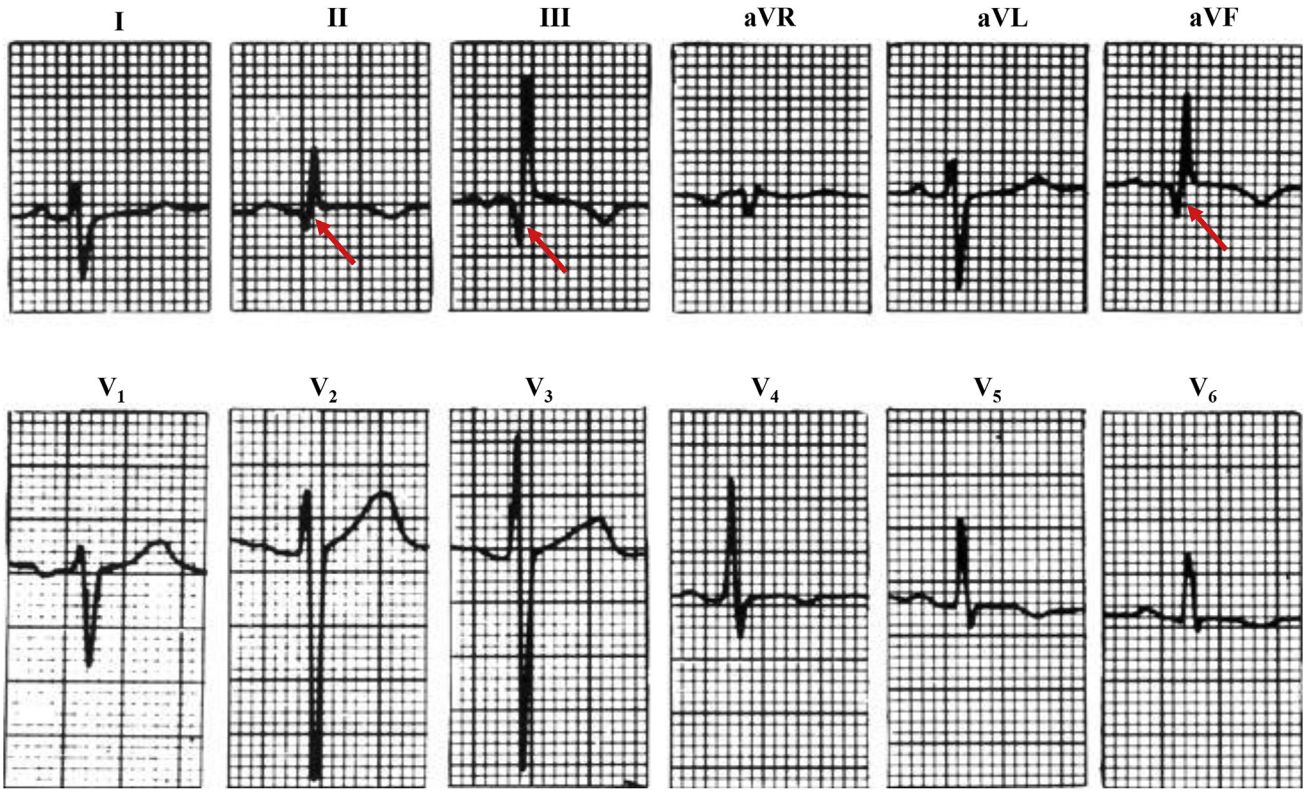


Fig. 3. QRS axis +115°, I and aVL rS; III qR; II, III and aVF qR with inferoapical post-ischemic T wave; V_2–V_3 deep S waves. LPFB and inferior MI accounted for Q waves in leads II, III and aVF (arrows). However, the R-wave amplitude in these leads is increased after LPFB but decreased after inferior MI. The mean QRS axis in the FP was shifted toward the vertical in LPFB but little changed or slightly shifted away from the vertical in inferior MI.

large population only 8 patients had LPFB. In this study LPFB was defined as frontal QRS axis ≥120°, lead I rS configuration, leads II, III and aVF qR configuration, and no pathological Q waves in leads II, III, and aVF following Castellanos et al. [7,8]. We consider Castellano's criteria restrictive and not suitable, because the q waves in the inferior leads can be ≥ 40 ms in duration when associated with inferior myocardial infarction (MI).

The prevalence of LPFB in a large French population of 69,186 aircrew members examined for fitness assessment in an aeromedical center was 0.13% [9], compared with 1.25% for incomplete right bundle branch block (RBBB), 1.1% for LAFB, 0.46% for RBBB, 0.08% for left bundle branch block (LBBB), 0.03% for incomplete LBBB, while the prevalence of left septal fascicular block (LSFB) was not reported.

2.1. Possible causes

LPFB is the rarest of all intraventricular blocks, and extremely unusual when isolated. In the following, the association of the conduction block with clinical entities is described as reported in the publications from our literature review.

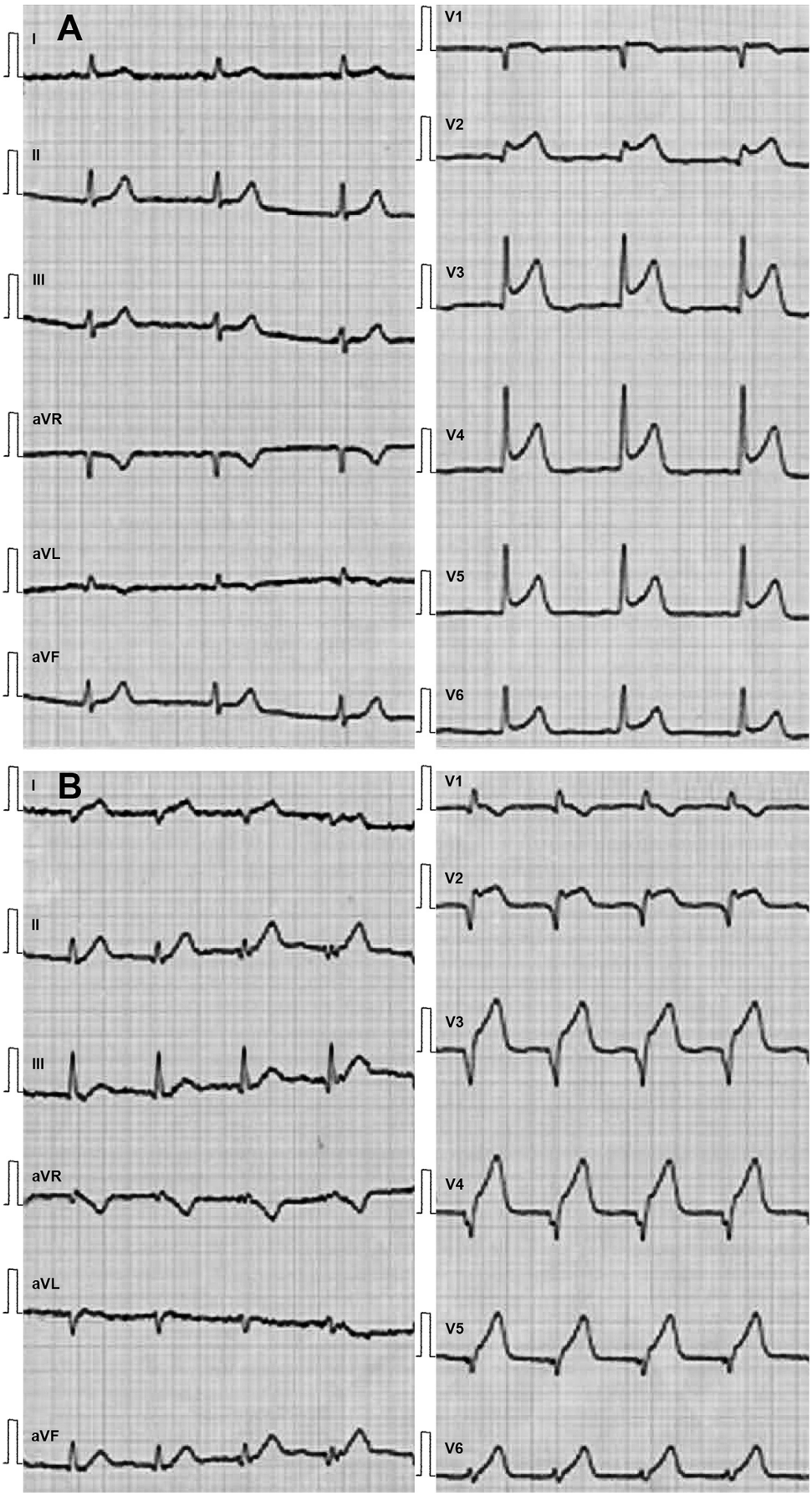
1) Coronary artery disease (CAD): Rizzon et al. [10] reported 15 cases with LPFB during acute MI. They observed that this ECG pattern was generally dependent on coexisting injury of the LSF and LPF. The anatomical reports did not confirm Rosenbaum's contention that LPFB was likely to be associated with more injury or extensive pathological abnormalities than LAFB [4,5]. The lesion associated with LPFB appeared to be less extensive and more discrete than those of LAFB. The LPF lesions were more proximal, any extension therefore being likely to involve the common LBB. It is worthy of note that chronic LPFB was frequently associated with calcifications of the mitral valve annulus and fibrotic lesions of the upper part of the atrioventricular junction.

Although rare, LPFB is a clinically important intraventricular conduction disturbance. Its appearance is reliably connected with inferior wall MI and generally reflects severe two- or three-vessel disease, requiring invasive investigation [11–13].

During the acute phase of ischemia in MI, LPFB is observed in ≈0.2–0.4% [14,15]. Association of LPFB and basal lateral (previously named dorsal) inferior MI accounted for Q waves (Q ≥ 40 ms) in the inferior leads II, III and aVF. However, the R-wave amplitude in these leads is increased in case of LPFB but decreased with inferolateral MI. Additionally, the QRS axis in the frontal plane (FP) shifts toward a vertical position in the presence of concomitant LPFB, but is little changed or slightly shifted away from the vertical in inferolateral MI. When inferolateral is accompanied by LPFB, there may be masking, imitation or enhancement of the effects of one lesion by the presence of the other [16]. A case of transient LPFB and various intraventricular conduction disturbances associated with acute anterolateral MI was reported by Ogawa et al. [17] Madias and Knez described transient LPFB during the treadmill stress test [18] (Fig. 2).

LPFB is rather frequent in inferior MI (5.5%). In the retrospective study from Godat et al., of a cohort of 830 patients referred for invasive investigation of suspected CAD, 163 patients had an old inferior MI. Nine patients (5.5%) showed a LPFB pattern; eight of these had three-vessel disease. The diagnosis of inferior MI had been made only in one case before entry of the patient into the hospital, since LPFB generally masks inferior MI [12] (Fig. 3).

Isolated LPFB is a very rare finding but the evidence of transient right axis deviation with a LPFB pattern was reported during acute anterior MI as related with significant RCA obstruction and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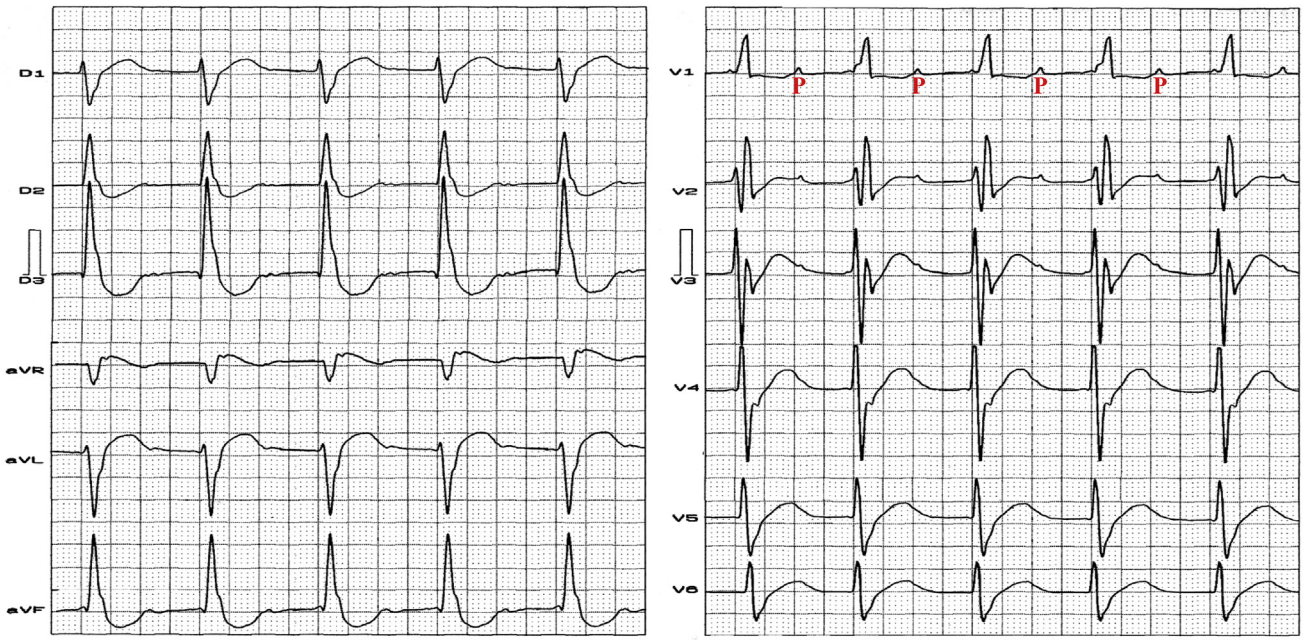

Fig. 5. Clinical features: 85-year-old female. **Autopsy diagnosis:** Lev's disease.
ECG diagnosis: 1st degree AV block + LPFB + RBBB: probable trifascicular block + digitalis effect.

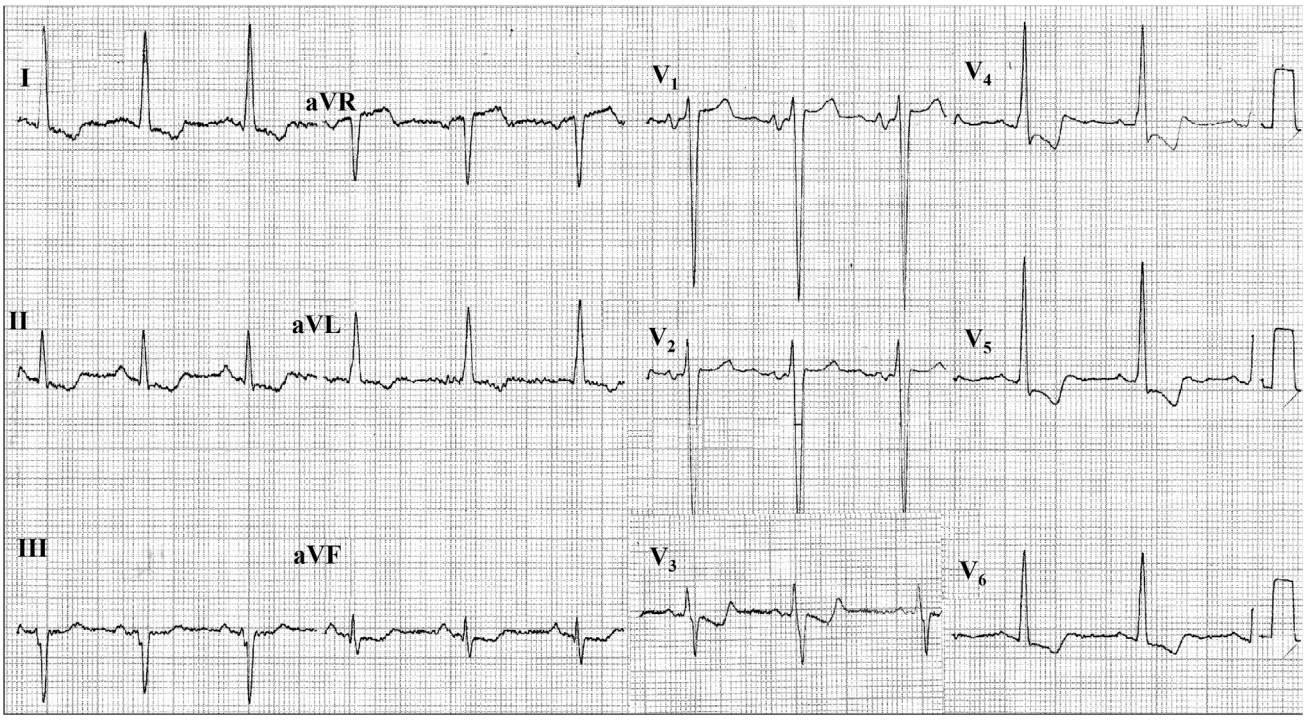

Fig. 6. ECG performed before the percutaneous septal ablation.
Clinical diagnosis: obstructive hypertrophic cardiomyopathy with a gradient in LVOT of 80 mmHg and NYHA IV, even with medication.
ECG diagnosis: left atrial enlargement + left ventricular hypertrophy (LVH) with strain.

collateral circulation between the left coronary system and the posterior descending artery. Patanè et al. presented a case of transient right axis deviation, transient LPFB pattern and transient junctional rhythm with acute MI and a significant LAD stenosis [15] (Fig. 4).

LPFB associated with Prinzmetal angina: Ortega-Carnicer

Fig. 4. A: Upwards concave STE followed by positive T waves across the precordial leads (hyperacute phase of anterolateral MI).
B: Right axis deviation, LPFB and a QS pattern from V_2 to V_6 suggesting transmural MI, higher precordial STE and qR pattern followed of negative T wave suggesting RBBB associated.

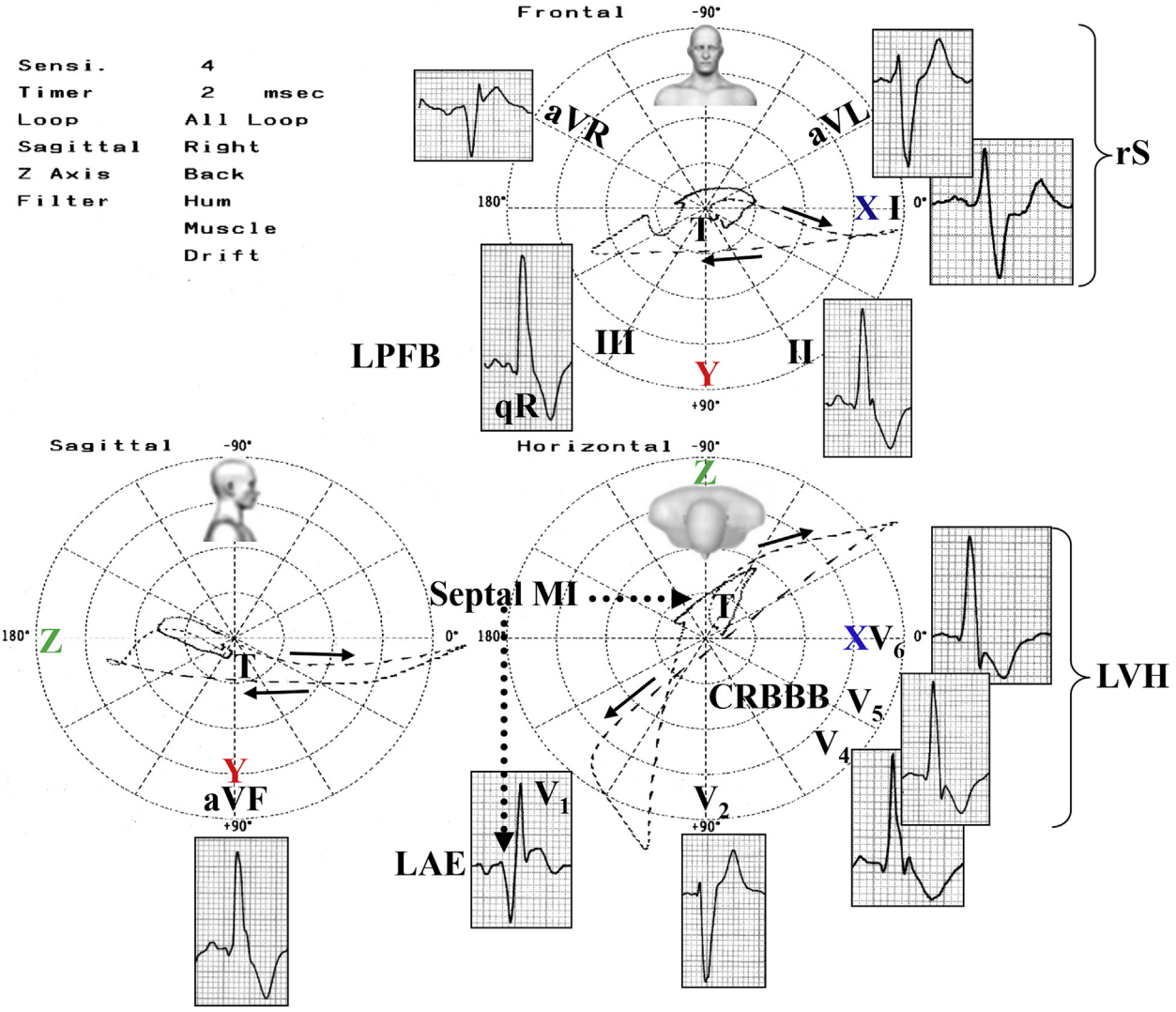

Fig. 7. ECG/VCG correlation immediately after percutaneous procedure.
ECG/VCG: LPFB + acute septal MI + LVH + RBBB.

et al. presented a case of a 49-year-old man with transient LPFB during Prinzmetal's angina with STE in the inferior leads; subsequently LPFB reappeared associated with acute inferior MI. These authors believe that the very early changes in the QRS axis and ST segment are explained by three important changes in leads overlying the affected area: 1) the conspicuous STE associated with reciprocal changes; 2) the "injury"-related intramural (local) block; and 3) the increase of the R-wave voltage, also dependent of dromotropic disturbance caused by the "injury" [19]. Note: This prominent R wave could explain the LSFB observed in the proximal obstruction of the LAD before its first septal perforator branch.

2) Familial progressive cardiac conduction defect or Lenègre disease [20]**:** Familial progressive cardiac conduction defect (PCCD) is a conduction disorder, that may progress to complete heart block. Affected individuals may be asymptomatic, or the condition may cause shortness of breath, dizziness, fainting, abdominal pain, heart failure, or sudden death. Mutations in several genes, including the *SCN5A, SCN1B* and *TRPM4* genes, can cause PCCD. Mutations in the *SCN5A* gene are potential etiologic factors for a number of overlapping syndromes [21]. Several other genes may be the cause when PCCD occurs with congenital heart disease. Familial PCCD is usually inherited in an autosomal dominant manner. However, not all individuals with the mutated gene will manifest PCCD; in those that do, symptoms and severity vary (reduced penetrance and variable expressivity). Autosomal recessive inheritance and sporadic cases have been reported as rare entities. Treatment includes permanent pacemaker implantation.

3) Lev's disease or progressive idiopathic sclerosis of the "cardiac skeleton": With a clinical behavior similar to Lenègre's disease, although typical for elderly patients. Both Lenègre's disease (known as progressive "primary" fibrosis of the His-Purkinje system) and the secondary mechanic injury, left-sided sclerosis of the "cardiac skeleton" or Lev's disease [22,23], cause intraventricular dromotropic disorders with QRS broadening into values $\geq$ 120 ms. Occasionally, they progress to more advanced (trifascicular) blocks, which may be expressed in the ECG as PR-interval prolongation with potential to cause sudden cardiac death by total trifascicular AV block.

Lenègre's and Lev's diseases are major indications for pacemaker implantation with an implantation rate of 0.15 per 1000 inhabitants per year. In Latin America the main cause is Chagas disease. The two entities, called PCCD, are inappropriately grouped as a single disease (Lev-Lenègre disease). However, Lenègre's disease is genetic and Lev's disease is degenerative. The last one is

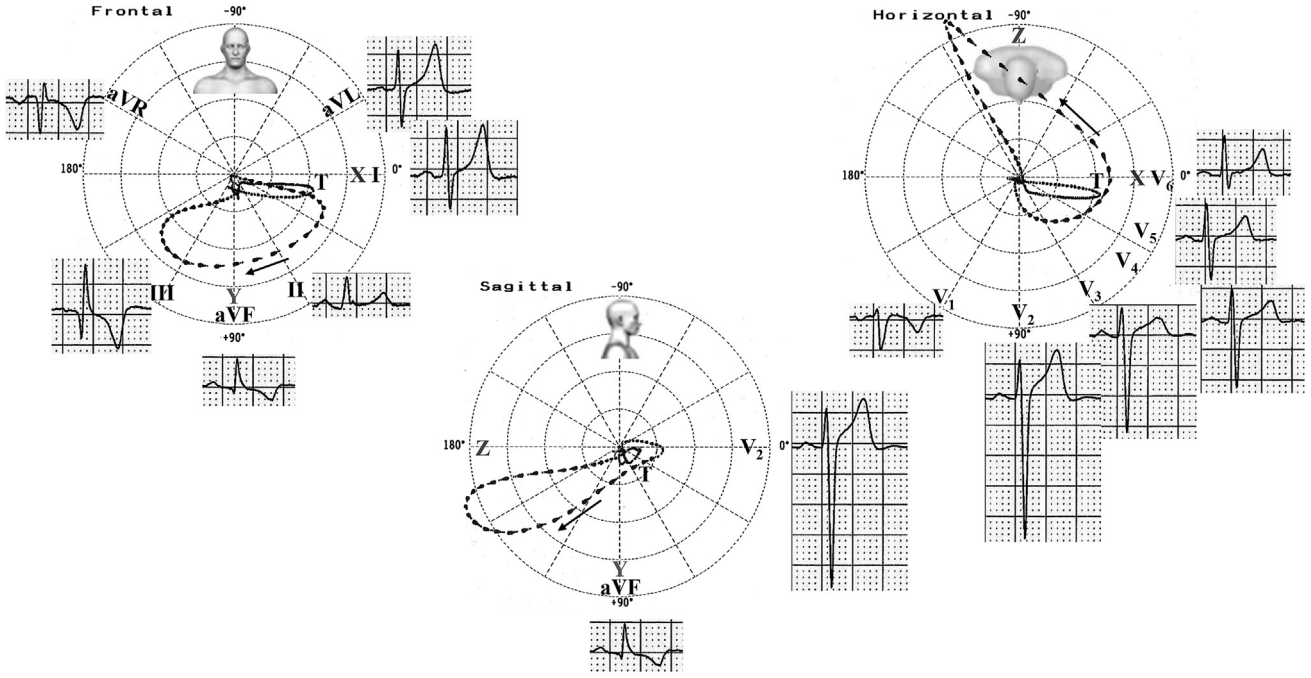

Fig. 8. VCG/ECG correlation in isolated LPFB.
FP-ECG: initial 10–20 ms vector heading upward and to the left; rS pattern in I and aVL; qR in inferior leads; RIII > RII; middle final notch in the descending limb of the R wave of lead III; QRS loop of clockwise (CW) rotation and broadened morphology.
Clinical absence of RVH, vertical heart or extensive lateral MI (the diagnosis of LPFB must obligatorily be clinical-electrocardiographic). A definite diagnosis should be made only in absence of RVH, "vertical heart" or lateral MI.
FP-VCG: Initial 10–20 ms vector heading above and to the left (near −45°) with delay (initial 10–25 ms); broad QRS loop, CW rotation; maximal vector ≈ +110°; almost all the QRS loop located below the X orthogonal lead predominantly in the inferior quadrants; QRS loop duration 110 ms; T loop with CW rotation, heading below and to the left near +10°.
HP-ECG: rS pattern in V_1 –V_2, deep S wave in V_2 (consequence of posterior dislocation and to the right of the final forces), scant progression of growth of R wave, transition zone in V_4 and Rs in V_6, prolonged R-wave peak time (≥50 ms) in V_5–V_6 and disappearance of q wave in these leads.
HP-VCG: Initial 10–20 ms vector heading to the front; QRS loop shape very similar to type C RVH; QRS loop of CCW rotation; greater area of QRS loop located in the left posterior quadrant; maximal vector of QRS around −80°; final portions with delay located in the right posterior quadrant (>20% of the QRS loop area located in the right posterior quadrant). T-loop minimally directed to the front and leftward (+10°) with CW rotation.
Right sagittal plane-VCG: Initial 10–20 ms vector heading to the front and above with delay; most of the QRS loop located in the inferoposterior quadrant; QRS loop of CW rotation; maximal vector around +160°; end QRS conduction delay; T loop heading to the front and below with CW rotation.
Conclusion: Isolated LPFB.

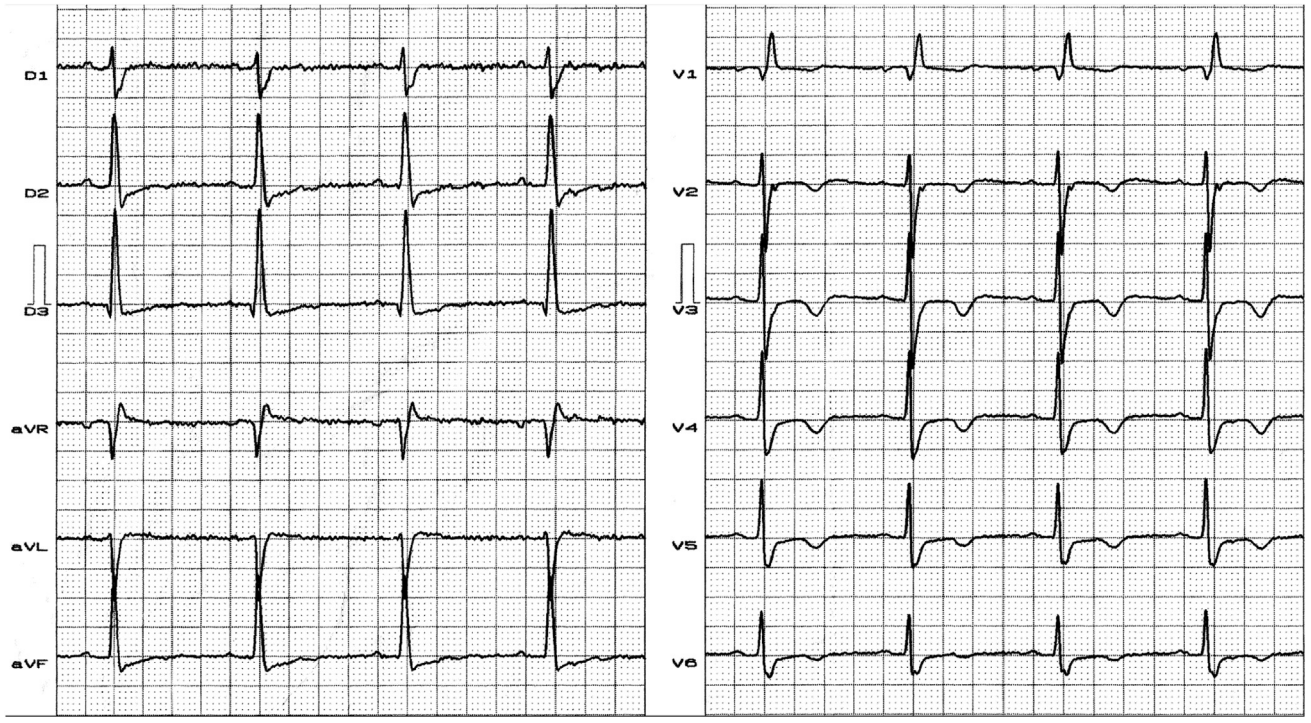

Fig. 9. Typical RBBB pattern associated with LPFB, septal infarction (qR in V1) and anterior ischemia.

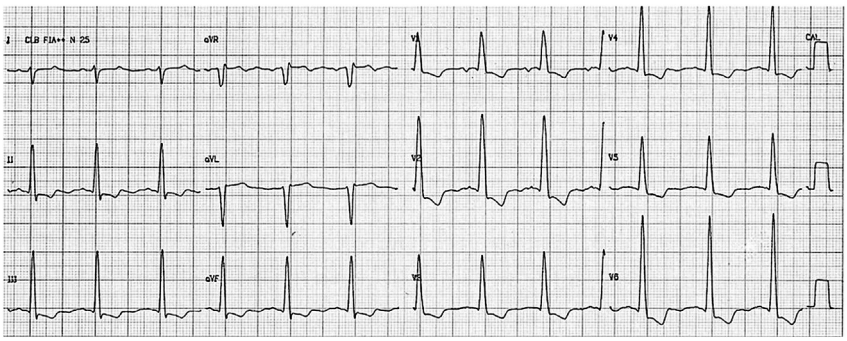

Fig. 10. Right axis deviation (QRS axis in +115°), rS pattern in leads I and aVL, tall R waves in lead III > RII, and qR pattern (embryonic q) in lead III. This ECG pattern in the limb leads, in the absence of clinical and echocardiographic evidence of RVH, lateral infarction and/or vertical heart associated with significant left ventricular hypertrophy, suggests LPFB. In the chest leads, prominent anterior QRS forces: R wave in $V_2 > 15$ mm. In the absence of RVH, laterobasal MI, RBBB, pre-excitation with a posterior anomalous pathway, hypertrophic cardiomyopathy, endomyocardial fibrosis, muscular dystrophy, and dextroposition, strongly suggests LSFB.

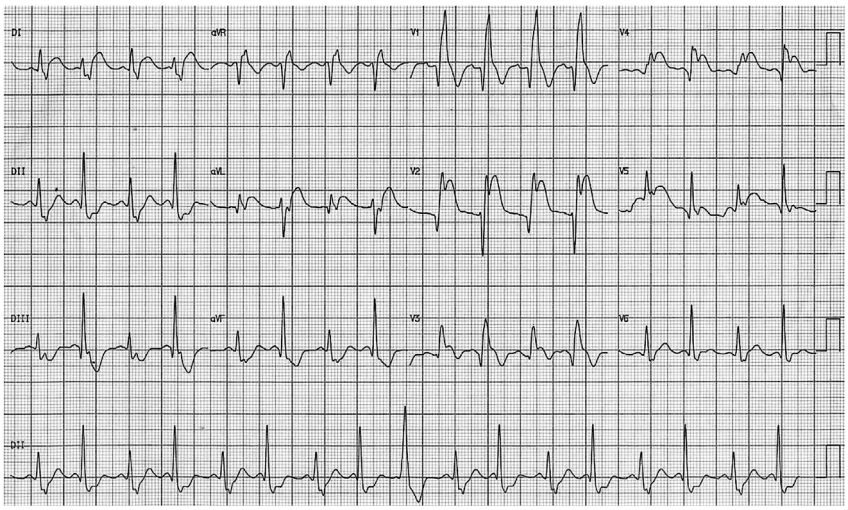

Fig. 11. Admission ECG.
Sinus tachycardia, rate-independent transient alternating LPFB associated with complete RBBB (qR pattern from V_1–V_4) + acute extensive anterior STEMI, caused by occlusion of the LAD after the first septal perforator and before the first diagonal branch of the LAD, because the injury vector points to the left leads (I and aVL) with reciprocal ST depression in the inferior leads. In precordial leads, the injury vector points to the front and minimally leftward, causing STE from V_2–V_5 (Fig. 12).

observed in elderly and is characterized by progressive mechanic fibrosis of the left cardiac "skeleton", mitral ring, central fibrous body, membranous part of the base of the aorta and apical calcification of the muscular septum. On the other hand, Lenègre's disease has a genetic background and occurs in younger people.

In Lev's disease, the fibrosis and calcification may involve the intraventricular His system, causing LBBB or RBBB associated with fascicular blocks [24].

Fig. 5 shows a typical ECG example of Lev's disease.

4) Chronic aortic regurgitation, aortic stenosis or both: Dromotropic disturbance is attributed to the mechanical effect of jet regurgitation on the posterior portion of the left septum, the site where the thick LPF traverses the LVIT. Among 304 cases of aortic valvulopathies studied for surgical selection, Marchandise et al. found a high incidence of conduction disturbances (16% in aortic stenosis and 18.4% in aortic regurgitation). The incidence of conduction disturbances increases with age, valvular calcifications, left ventricular hypertrophy with strain pattern, heart failure, and CAD [25].

5) Chronic chagasic myocarditis: From a large population database of primary care patients (7590 cases), LPFB was observed in 55 individuals (0.72%) [26].

6)Acute myocarditis [27].

7)Infiltrative cardiomyopathies: cardiac amyloidosis [28], sarcoidosis [29], hemochromatosis cardiomyopathy, and Fabry disease.

8) Severe obstructive hypertrophic cardiomyopathy treated with percutaneous alcohol ablation: in the procedure, reduction of the hypertrophic septum is done by absolute alcohol injection in the first septal perforator branch of the LAD. Figs. 6 and 7 illustrate the ECG consequences of the procedure.

9)Systemic hypertension [30].

10)Interventricular septal tumor [31].

11)Hyperkalemia: in this case disappeared promptly by hemodialysis, as the serum potassium level returned to normal [32].

12)Selective coronary arteriography in the RCA [33].

13)Acquired ventricular septal defect: in cases of inferior wall MI, complicated by rupture of the inferior septum, resulting in isolated LPFB [34].

14) Acute pulmonary embolism? [35] Although described as LPFB by Scott, the diagnosis of LPFB should always consider the clinical aspects. LPFB diagnosis is not possible in the presence of right ventricular hypertrophy (RVH).

15) Hereditary: Pseudo LPFB? [36] Hereditary right axis deviation with pseudo LPFB and incomplete RBBB resulting in right axis deviation was described in a family. Clinical, radiological and

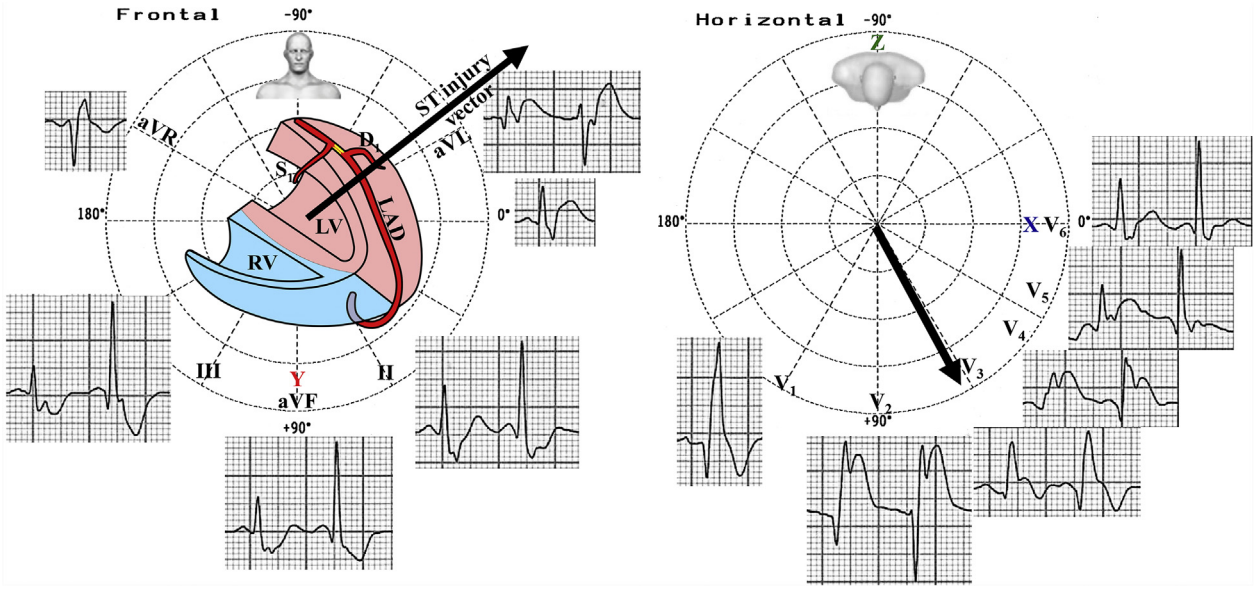

Fig. 12. ECG/VCG correlation in the FP and HP.
FP: ST injury vector directed to left and upward (ST segment elevation in I and aVL and ST depression in III, aVF and II).
HP: St injury vector directed to front and leftward near +60°. ST segment elevation from V2 to V5, qR from V1–V4 (anterior MI complicated with RBBB).

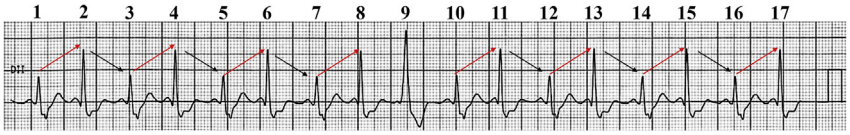

Fig. 13. Long II lead. Beats 1, 3, 5, 7, 10, 12, 14, and 16 have a RBBB pattern; beats 2, 3, 4, 8, 11, 13, 15 and 17 have a minimal degree of LPFB + RBBB; beat 9 has a major degree of LPFB without RBBB.

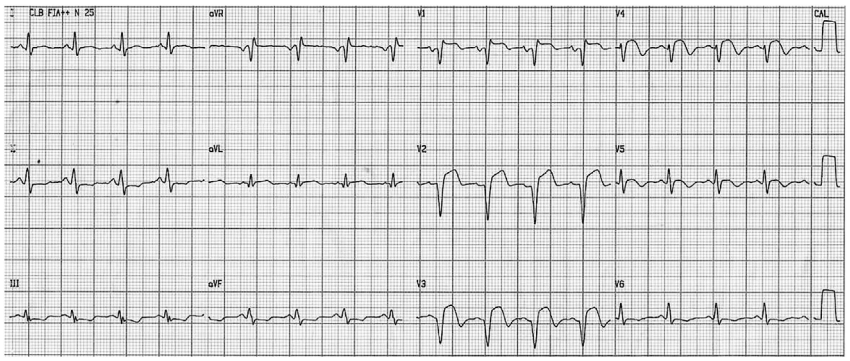

Fig. 14. ECG after percutaneous coronary intervention, RBBB disappears, anterior transmural MI, ST segment elevation from V1 to V4, negative T wave in left precordial leads.

echocardiographic evaluation excluded structural and functional cardiac abnormalities as well as chest deformities and lung disease [36].

2.2. LPFB classification

I. Isolated or associated with other blocks.

a) **Isolated LPFB:** unusual (Fig. 8).
b) **Associated with RBBB:** more frequent [37–40] (Fig. 9).

Demoulin et al. [41] reported 10 cases with histopathologic correlates of LPFB, two isolated cases and eight associated with RBBB. Among these eight cases, LPFB was intermittent in one and alternated with LAFB in two cases. Severe pathologic changes in the conduction system were found in all but one case, where LPFB resulted from a very recent MI. The lesions almost exclusively involved the LAF and LSF, where they actually predominated.

c) **Associated with LSFB:** only one case was described in the literature [42] (Fig. 10).

II. According to steadiness:

a) **Permanent:** it is the most frequent and always complete (QRS duration ≥120 ms).
b) **Intermittent or of second degree LPFB** that could be:

Table 1
ECG/VCG differential diagnosis between isolated LPFB and LPFB associated with CRBBB in the frontal plane.

	Isolated LPFB	LPFB + CRBBB
QRS duration	90–110 ms	≥120 ms
Location of the QRS loop	≥ 40% of the area located in the left inferior quadrant	≥ 40% of the area located in the right inferior quadrant
Vector of final 20 ms	There might be delay, but discrete.	With important delay to the right.
Number of comets in the QRS loop (if 1 comet = 2 ms)	45 to 50	≥60

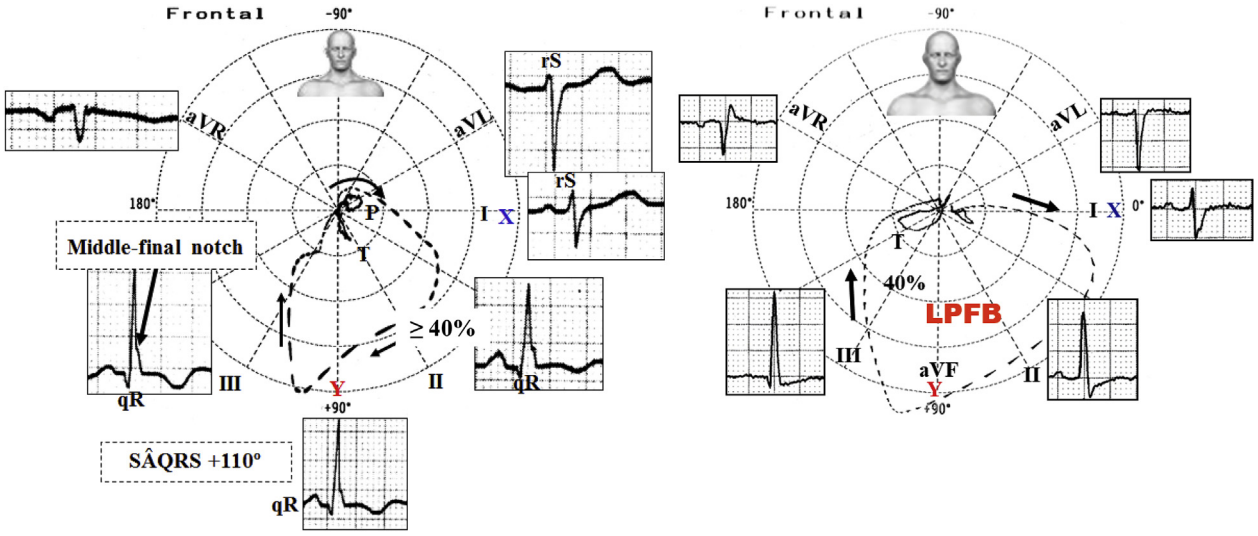

Fig. 15. ECG/VCG correlation in case of isolated LPFB (A) and LPFB associated with RBBB (B).
Horizontal Plane: QRS loop very similar to RVH of type C [50]; QRS loop of CCW rotation. The rotation could be in "eight"; initial 10–20 ms vector heading to the front and the right or left; greater area of QRS loop located in the left posterior quadrant; maximal vector of QRS around −60 to −110°; final portions with delay (60–100 ms) and located in the right posterior quadrant; 20% or more of the QRS loop area located in the right posterior quadrant; T loop located to the front and the left (+60°) and CW rotation.

- **Rate-dependent LPFB:** tachycardia-dependent or in "phase 3", and bradycardia-dependent or in "phase 4".
- **Rate-independent LPFB.** Mechanism: Mobitz type I; Mobitz type II by Wenckebach phenomenon; and by significant hypopolarization (Figs. 11–13).

Primary percutaneous coronary intervention with stent implantation in the LAD showed total occlusion in the middle portion of the LAD. ECG performed immediately after stent implantation (Fig. 14).

Complicated evolution of the procedure, because there was no myocardial reperfusion (epicardial reperfusion without microcircular reperfusion). This phenomenon suggests microcirculation injury, no washout of metabolites, and therefore the pH is very low (tissue acidosis). The hydrogen, calcium, potassium, and detritus are accumulated in this area. In addition, transient LPFB and RBBB disappear after stent implantation, QRS width decreases, Q waves in aVL, V_1–V_3, STE in the anteroseptal wall.

2.3. ECG criteria of LPFB in the FP

QRS deviation to the right (QRS axis ≈+120° between +80 and +140°); with SIQIII pattern, rS pattern in leads I and aVL; qR pattern in III, aVF and II: Q wave is always present in III and may be small or absent in II or aVF; characteristic notch in the descending limb of the R wave in III (middle-final notch); RIII > RII: QRS axis closer to +120° (III) than +60° (II); when closer to the latter, it would indicate an incomplete LPFB. q wave in III is always greater than q wave in II and aVF. If there is association with inferior MI, Q wave >40 ms [43]; QRS duration <120 ms if isolated (without RBBB); R-peak time in aVF ≤35 ms [44], and the PR interval is prolonged [45].

2.3.1. ECG criteria in the HP

V_1 and V_2: rS pattern or rarely QS; S wave of V_2 -V_3 very deep as a consequence of posterior dislocation and to the right of the terminal QRS forces; scant progression of R waves growth in the precordial leads. Normally the R wave amplitude increases from V_1 to V_5. Around V_3, R waves become larger than S waves and this is called the 'transitional zone". In LPFB, the transitional zone is dislocated to the left in V_5 and V_6: Rs pattern; prolonged R-wave peak time in V_5–V_6 (≥50 ms) and disappearance of q wave in these leads.

2.4. VCG criteria of LPFB

FP: Vector of initial 10–20 ms heading above and to the left (near −45°) with possible delay (initial 10–25 ms) [46]. If associated to inferior MI, superior initial forces of 25 ms or more (more than 12.5 dashes above the orthogonal X lead. 1 dash = 2 ms) [45]; broad QRS loop, with CW rotation. Cooksey, Dunn and Massie wrote that occasionally, it may be in "eight" with a CCW rotation in the terminal portion (10%) [47,48]; maximal vector near +110° (+80° to +140°) [49]; almost all the loop is located below the X line (0 to ±180°) in the inferior quadrants; 20% of the loop located in the right inferior quadrant. If there is association to complete RBBB, 40% or more [37,39,40]; afferent limb heading below and slightly to the left, and the efferent one to the right; middle-terminal portion of the QRS loop (vector of 60–100 ms) with delay. It may possibly reach the right superior quadrant; QRS loop duration up to 110 ms if isolated. In association with complete RBBB >120 ms; normal ST-T vectors in isolated LPFB: T loop with CW rotation, heading below

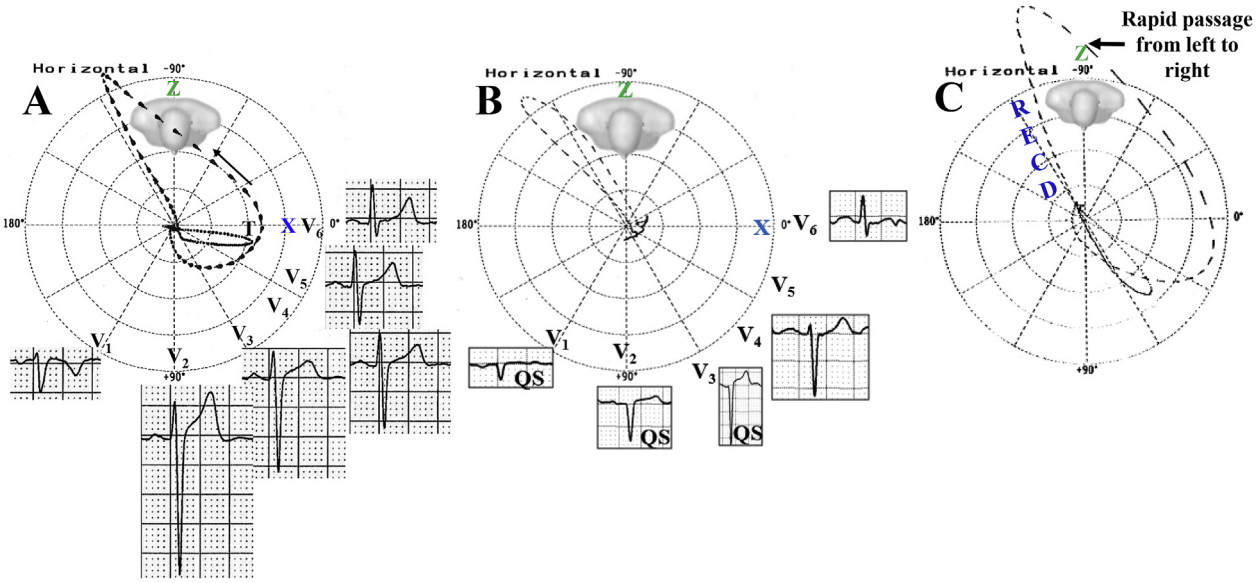

Fig. 16. A: Isolated LPFB in the HP; B: Type C RVH in the HP in a man with severe COPD; C: right posterior subdivision block in a healthy individual.

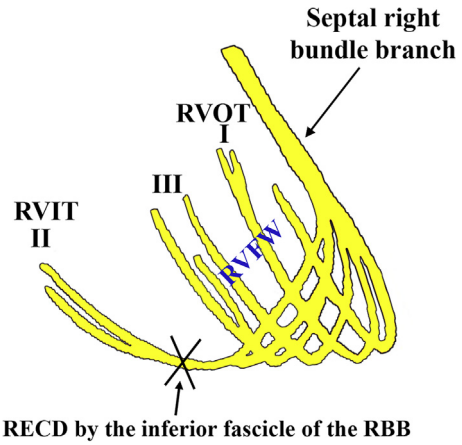

Fig. 17. Outline of the inferior divisional block in RVIT (RECD type II) [23,55,56]. RVIT: right ventricular inflow tract; RVOT: right ventricular outflow tract; RVFW: right ventricular free wall.

and to the left. If in association with complete RBBB: alteration secondary to ventricular repolarization.

Table 1 and Fig. 15 show the differential diagnosis between isolated LPFB and LPFB associated with CRBBB.

2.5. LPFB differential diagnosis

Obligatorily, the diagnosis of LPFB must be clinical-ECG-VCG. A firm diagnosis is not possible in the presence of: a vertical heart in slender subjects (ectomorphic biotype); presence of any cause for RVH, especially chronic obstructive pulmonary disease/emphysema; large lateral wall MI: QS in I and aVL [51].

Right end conduction delay (RECD) by the inferior fascicle of the RBB or RECD type II of our classification [52]: characterized by presenting ECD located in the right inferior quadrant in the territory of the inferior fascicle of the right branch. It corresponds to the territory of the right inferior fascicle (Fig. 17). The differential diagnosis occurs with LPFB. Many of the cases described in literature as LPFB are, the way we see it, RECD type II, and since their ECG/VCG differences are very subtle, the diagnosis must always be clinical-ECG-VCG.

In the horizontal plane, it is hard to differentiate isolated LPFB from RVH VCG type [53] and right posterior subdivision block [54] (Fig. 16).

2.6. Differential diagnosis between right posterior subdivision block, segmental blocks of the right bundle-branch or RECD type II and LPFB

RECD are ECG/VCG changes, secondary to physiological delay or to true dromotropic disorders in the territory of one of the hypothetical three fascicles or actually, fiber contingents of the RBB on the free wall of the RV, in isolation in the right ventricular free wall. In case of block, there should be a dromotropic disorder or slowing of the ventricular activation process. These blocks cause localized or regional delay in the basal portion of the RV free wall. Zonal right ventricular blocks correspond to block of the superoanterior division of the right bundle on RV free wall (on RVOT) or inferoposterior zone (on RVIT) of the RV free wall. Fig. 18 shows hypothetical fascicles (or contingents) of the RBB in the RV free wall.

Right distal peripheral blocks generally do not hide the electrical signs of a non-activatable myocardium [57]. These blocks enhance the manifestations of later occurring electromotive forces resulting from RV activation. Therefore, they do not interfere with the development of ventricular electromotive forces in the zones affected by myocardial injury. This happens both in the presence of a right anterior subdivision block, which increases the duration of basal electromotive forces by at least 10 ms, that normally develop in 64–72 ms [58] and in the presence of a right posterior subdivision block. This latter block prolongs the electromotive forces originating in the posterior and medial and inferior posterolateral septal regions of the right ventricular free wall by an average of 12 ms, which become activated in 30–45 ms [59].

Table 2 shows the differential diagnosis between right posterior subdivision block and LPFB.

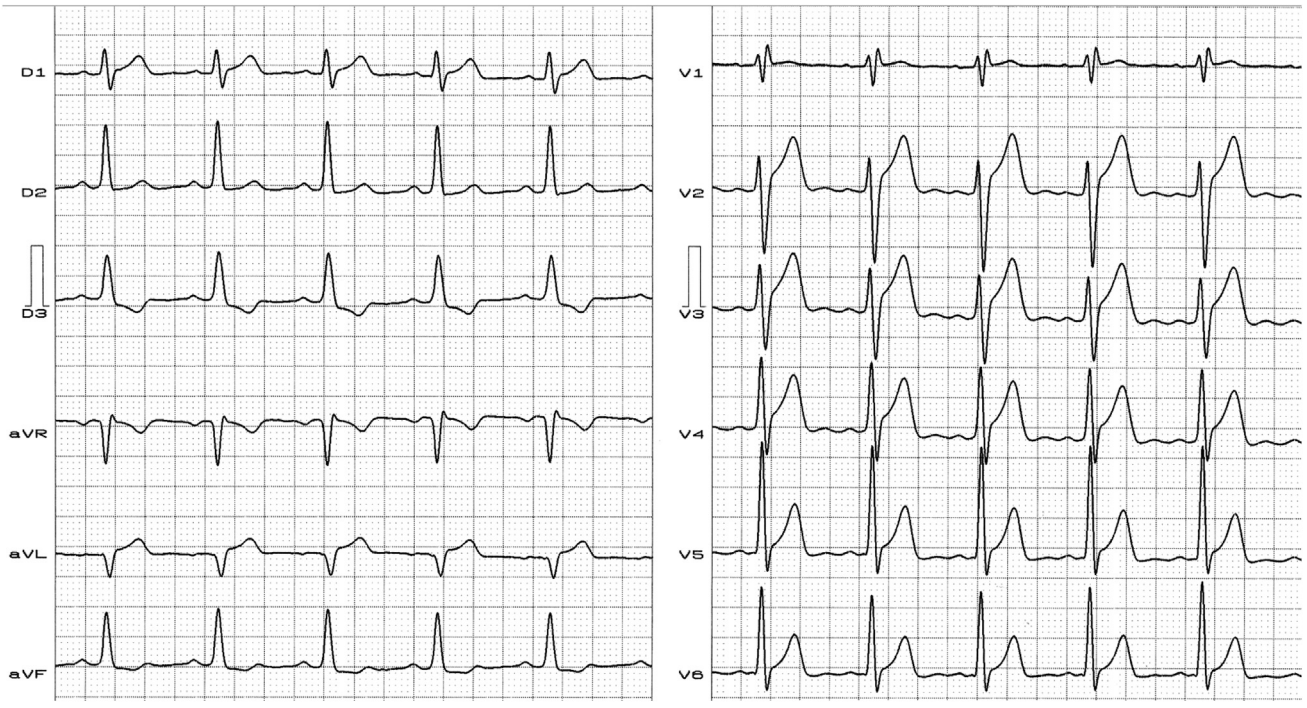

Fig. 18. ECG of a young male athlete with normal heart.
Clinical diagnosis: Healthy patient requesting examinations to assess his eligibility for competitive sport.
ECG diagnosis: SÂQRS: + 85°. RII > RIII. SAT: + 5° to the front and the left. Morphology of incomplete RBBB: rSR′ in V_1.
Conclusion: ECG of RECD type II. Why? Because of the patterns on ECG/VCG correlation in the FP (Fig. 19).

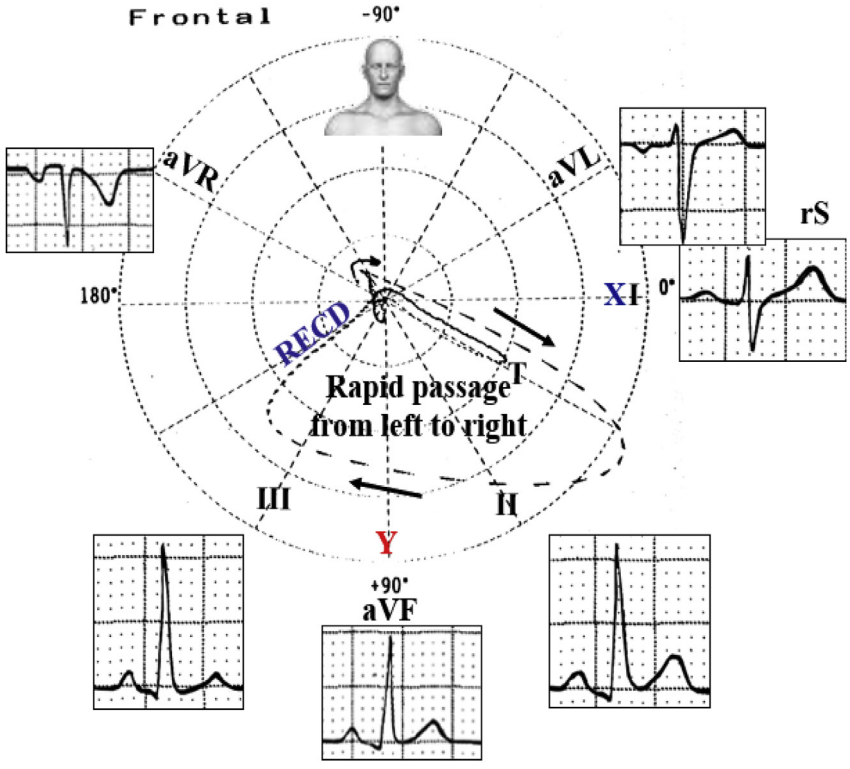

Fig. 19. ECG/VCG correlation: blockage of the inferior fascicle of the RBB.
RECD type II: RECD located in the right inferior quadrant in the territory of the inferior fascicle of the right bundle branch. SÂQRS: +95°. SI-RII-RIII pattern (RIII <15 mm). I and aVL: rS. II and III: qR. The descending ramp of R wave is slightly slow. It may present differential diagnosis with LPFB.

Table 2
Differential diagnosis between right posterior subdivision block (RECD type II) and LPFB.

	RECD type II or right posterior subdivision block	LPFB
PR interval	Normal	Frequent prolongation
Association with inferior infarction	No	Frequent
Voltage of RII and RIII	≤10 mm	≥15 mm
RII/RIII voltage ratio	RII > RIII	RIII > RII
R-wave peak time in aVF, V_5– V_6	Normal	Increased: up to 30 ms
R-wave peak time in aVL	Normal	Decreased: up to 15 ms
Aspect of QRS loop in the FP	CW and with characteristic rapid passage from left to right between 30 and 50 ms	CW, aspect of "fat" loop and maximal vector near +120°
Clinical factors that should be excluded	Not stated	Vertical heart, RVH, emphysema, COPD and lateral infarction

RECD: right end conduction delay; LPFB: left posterior fascicular block; CWR: clockwise; FP: frontal plane; RVH: right ventricular hypertrophy; COPD: chronic obstructive pulmonary disease.

2.7. Clinical significance of RECD

In 90% of cases they represent normal variants. The clinical significance and interest lie in the fact that:

1) They may be confused with LAFB and LPFB [52];
2) They may be confused with electrically inactive areas in the anterior and the inferior walls [60];
3) They may represent the ECG/VCG pattern of the Brugada syndrome [61].

Fig. 18 shows a typical example of the inferior fascicle of the RBB or RECD type II of our classification. This dromotropic disturbance has differential diagnosis with LPFB.

3. Conclusion

Isolated LPFB is an extremely rare finding both in the general population and in specific patient groups and the QRS loop in the HP is indistinguishable from the QRS loop of the RVH type C characteristic of COPD. In isolated LPFB 20% of the QRS loop is located in the right inferior quadrant and when associated with RBBB ≥40%.

In our experience, isolated LPFB is much more unusual than LSFB. The diagnosis of LPFB should always consider the clinical aspects, because a definite diagnosis cannot be made in the presence of RVH (COPD/emphysema), extensive lateral MI or extremely vertical heart.

Intermittent LPFBs are never complete blocks and even in the permanent ones, one cannot be sure that they are complete. When LPFB is associated with RBBB and acute inferior MI, PR interval prolongation is almost constant.

Conflicts of interest

None.

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